EXHIBIT 34

the extraordinary decision to retrofit its breathing machines with 3-D printed plastic tubing that allowed doctors to ventilate two patients simultaneously, using the same device.[6]

I'd completed my medical training in New York City as a resident in internal medicine twenty years earlier, and I went to medical school at Mount Sinai Hospital in Manhattan. Among my most vivid memories from training were covering the medical floors at Elmhurst Hospital in Queens. The hospital was located in one of the most ethnically diverse neighborhoods in the country, and the community's rich culture deepened the complexity, and gratification, of practicing medicine there. I knew the hospital's capable staff, and its immense capacity. Watching the scenes unfold—of Elmhurst Hospital being overrun with COVID patients, of refrigerator trucks parked outside, and of doctors and nurses describing their harrowing experiences—was hard to bear.

It was stunning, and it was shocking. But above all, it was terrifying. What my medical colleagues in the city described to me again and again was pervasive fear: Fear that they could spread the virus to their families, as each day New York hospitals were using as many masks, gloves, and gowns as they normally consumed in an entire month during usual times, quickly draining whatever stockpiles they had. Fear that they didn't know how to care properly for the sick patients overwhelming their wards, suffering from a virus that nobody yet understood. Fear that they couldn't predict how or when the arc of infection would start to ebb. And fear that a lot of lives would be lost.

It was a harrowing epidemic that brought the city's vaunted healthcare system much closer to the brink of collapse than most people, even now, recognize.

Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2 or SARS-2) is the virus that causes the disease that we've come to know as COVID-19. By the time the first cases of community spread were diagnosed in late February, SARS-CoV-2 had already rooted itself in our communities. It had been here for a while, at least since January, replicating, spreading, and doubling its numbers every two to three days.[7] Then, in March, after thousands of cases had accumulated, the virus abruptly burst into public view.

The virus didn't arrive with a group of visitors from China, where it originated, or from Italy, where it established its next major foothold. Instead, it likely rode along the breath of probably hundreds of different travelers from a variety of locations, each ferrying the infection, and evading the porous controls that the federal government had put in place at US airports. At the time nobody knew what was happening. Nobody knew how much virus was being carried by people who showed no outward symptoms of the disease. These were people who might never manifest any signs of illness but were still contagious. Without the ability to test people for the virus, we had no way of detecting its spread. We certainly had no way of stopping it.

This wasn't because the United States had never imagined it might fall victim to a deadly pandemic. We certainly had imagined the possibility. In some respects, we had been preparing for this moment through three presidential administrations, starting with George W. Bush, who warned in a 2005 speech, following the outbreak of Severe Acute Respiratory Syndrome, or SARS-1, and then avian flu, that "scientists and doctors cannot tell us where or when the next pandemic will strike, or how severe it will be, but most agree: At some point, we are likely to face another pandemic. . . . Our country has been given fair warning of this danger to our homeland and time to prepare."[8] We had a pandemic playbook on the shelf, ran exercises simulating the threat countless times, and developed the Strategic National Stockpile to store the medical countermeasures that the top experts thought the country would need. But when the pandemic we long feared finally arrived, we weren't ready. Many of the plans and preparations turned out to be a technocratic illusion. The stockpile lacked key essentials. A lot of what it contained didn't work. It was a metaphor for our fragile response.

When I worked in the federal government in public health roles,

we would say that planning for medical calamities provides you with no assurance that you're prepared to deal with one. That was certainly true for COVID. The US never developed a pandemic strategy that would be broadly relevant to a range of predictable and unexpected viral threats, and the country was slow to realize the ways in which the plan we had created and tried to work from, which focused almost exclusively on the risk from flu, wouldn't apply to COVID. The federal government started off in a weak position, with plans that were ill suited to countering a coronavirus. This mismatch between the scenarios we drilled for and the reality that we faced left us unprepared. Poor execution turned it into a public health tragedy.

It was an alarming state of vulnerability for a country with the world's most technologically advanced healthcare system. Owing to mistakes in how we deployed diagnostic tests for COVID, we left ourselves blind to the virus and allowed it to spread widely and largely unchecked, so we were never able to trace its early spread and contain it. Even when the shortcomings became obvious, the Centers for Disease Control and Prevention (CDC) continued to rely on its systems for monitoring and responding to influenza, insisting even into 2021 that its flu-based interventions were the right tools in the fight against COVID. We didn't pursue an approach that closely tied our efforts to track and contain the spread of SARS-CoV-2 to the characteristics of the virus. This central shortcoming explains many of the gaps in our response to the pandemic we actually faced.

Yet COVID shouldn't have been such a surprise. There had been earlier outbreaks of new and deadly strains of coronavirus. COVID's close cousin, SARS-1, appeared in 2002 and spread threateningly in 2003, and another dangerous coronavirus, the Middle East Respiratory Syndrome, or MERS, emerged ten years later. Moreover, the scientific literature over the last decade is riddled with reports of SARS-like coronaviruses that were found in bats and appeared to have the potential to sicken humans.

In 2012, six people developed an illness with symptoms match-

an unexpected event, yet it wasn't properly anticipated. The weaknesses and failures it revealed cost America dearly.

In one of the largest studies done to estimate the number of New Yorkers stricken with coronavirus at the height of the city's epidemic, researchers found that between February and July 2020, about 1.7 million people were infected. About 1 percent of them died.[25] During that dreadful spring wave of infection, *The New York Times* described "an apocalyptic coronavirus surge" of victims streaming into the city's hospitals.[26] The winter was even worse for the country, when the epidemic widened. Yet at the same time, on many days, the nations of China, Japan, South Korea, Taiwan, Singapore, Hong Kong, Thailand, Malaysia, Vietnam, New Zealand, and Australia were collectively registering fewer daily COVID cases than the city of Los Angeles.[27] Some of these nations, especially China, had employed draconian tactics that would have been firmly rejected in the US. But others had used mostly wide-scale testing and tracing to contain their spread. The Pacific Rim had figured out how to control the virus. We had not.

The pandemic devastated New York and left America with a palpable sense of vulnerability. To those who suffered and died, and to the rising generation in our country, we owe a clear-eyed review of the facts. What did we get right and wrong? What did we miss and why? What can we build on, and what must we build anew? What should we have known at the outset, and what must we never forget? These are the questions that guide this book. And along with them, undiminished by my effort to provide a comprehensive accounting of what went wrong, is a sense of awe at America's capacity to rise to a challenge, our innovation and ability to quickly develop highly effective drugs and vaccines, and the willingness of men and women in all walks of life in our society to take on terrible burdens for the sake of helping others.

Those NYU medical students pressed into emergency service, and in whose shoes I walked two decades ago, were certainly scared in the early days of pandemic. But they showed up. Like the doc-

fort to conceal its spread. In fact, it was his reporting that broke the story—despite China's initial denial—that there were cases of SARS-1 spreading in the northern parts of the country.[7] Even though SARS-1 was largely confined to a handful of regions, it had wrought havoc in some of the world's financial capitals. It was proof of the kind of severe economic and social dislocation that a dangerous epidemic could cause. The new coronavirus looked as if it could be an order of magnitude worse. It had Pottinger very worried.

During the opening weeks of the COVID outbreak, Pottinger was in touch with doctors he still knew inside China, and the firsthand accounts he was getting were radically different from what the White House was hearing from Beijing and the WHO. The Chinese doctors told him that the virus was circulating in the community and was already spreading in several provinces. One doctor believed that at least half the cases in Wuhan were people who were entirely asymptomatic. This was a respiratory epidemic driven by invisible cases, the doctor told Pottinger. Forget 2003, the doctor said. This was 1918. Pottinger went into the Oval Office and told the president everything he had heard from his Chinese sources. There were more than 23,000 people arriving each day from China, Pottinger said; the US needed to tap the brakes.[8] Two days later Trump announced that he would suspend most flights arriving in the US from China. By this point, these measures might slow the pace of introductions in the US and buy us some time, but the novel coronavirus had already arrived. The travel restrictions wouldn't prevent a US epidemic.

Early on, Pottinger also raised concerns about potential shortages of medical supplies like masks. But not everyone was similarly convinced of the dangers. As the pandemic later took its grip on the nation, he was one of the rare White House officials who routinely wore a mask to work, despite persistent scorn by colleagues, who labeled him an alarmist. Pottinger had two older relatives living with him at home and was so worried about the lax precautions being observed inside the White House that he had his office moved

from the West Wing to the Eisenhower Executive Office Building, an historic and ornate office building located next to the White House that housed many of the staff who supported the president. It was Pottinger's own act of social distancing. He was worried that he would catch the virus in the White House and bring it home with him.

Pottinger was also worried about continuity of government. He and O'Brien had decided that the two of them would remain physically separated even as they worked together. They would avoid face-to-face meetings, even when they were both inside the White House complex, to reduce the risk that they both could come down with the virus at the same time. O'Brien also backed Pottinger's risk assessment and issued an order requiring the staff of the National Security Council to wear masks. Pottinger worked with a Taiwanese official to secure half a million masks for the US, taking 3,600 for the staff of the NSC and the White House medical unit and donating the rest to the US Strategic National Stockpile.[9] Some in the White House were angered by Pottinger's action, worried about the optics of the White House staff acquiring surgical masks at a time when US officials were still discouraging the American public from buying and using their own masks. There was a shortage of medical masks in hospitals. But Pottinger's concern about the casual procedures at the White House would prove prescient; in the coming months the White House would be host to several outbreaks.

Pottinger knew that the risks were being compounded by our inability to test people for the virus. We had no reliable way of knowing how widely it might already be spreading. The US had the capacity to identify only a small number of infections, those with an obvious link to China that would qualify a patient to be tested under the CDC's narrow guidelines. The lack of testing created a false sense of security. If you look for cases only among people coming from Wuhan, you will find cases only among people coming from Wuhan. That's precisely what was happening.

Four days after the Washington State man was identified, a Chi-

cago woman was hospitalized for pneumonia and became the second person in the US to be diagnosed with COVID.[10] One day later, in California, a third case was uncovered. All three had recently traveled to Wuhan, and each recovered. The Chicago woman's husband became sick with COVID one week after she fell ill—the first documented case of person-to-person transmission of SARS-CoV-2 in the US. In Chicago, local health officials identified 372 people that the couple had been in contact with, and of those, 347 underwent active monitoring, including 195 healthcare personnel. No further cases were identified as a result of their illnesses.[11] In total, public health officials were monitoring sixty-three additional individuals in twenty-two states at that time. All were suspected of having COVID. Almost all had symptoms of respiratory infections, and all had some connection to Wuhan. Most were in some form of quarantine. Almost all of them would test negative for the coronavirus.[12]

Back in Washington, officials thought they were finding and isolating the virus and preventing new outbreaks.[13] Robert Redfield told the White House that the CDC's efforts were, so far, containing further spread. In fact, by January 25, the virus was very likely already being transmitted to ten new people every day in California alone, according to estimates made later by researchers at Northeastern University's Network Science Institute.[14] But during the critical months of January and February, nobody knew just how widely the virus was already spreading.

So public health officials were alarmed on February 28, when a second case emerged in Seattle, in a person who had no physical connection to the first Seattle-area patient, the one who had been diagnosed on January 20 after traveling from Wuhan. Using data that mapped the precise genetic sequence of the two coronavirus infections, scientists eventually found that the two viral strains, separated by more than a month, differed only by two small mutations. They looked connected.

Dr. Trevor Bedford, an associate professor in the Vaccine and Infectious Disease Division at the Fred Hutchinson Cancer Research Center in Seattle, was advancing the use of sequencing as a way to track the migration of coronavirus across the world. It was a relatively new field of science called genetic epidemiology, or "gen epi."[15] By sequencing the virus's RNA, he could trace the subtle ways its genetic code mutated as it passed from person to person, through successive generations of replication. Using the trail left by these small changes, he could track individual virus strains as they moved around the world. Coronavirus mutates more slowly than many other RNA viruses, so as it replicates and spreads, it does not collect as many changes. Still, it mutates about once every two weeks along a transmission chain. By looking at minor differences between the two Washington State strains—the one diagnosed on January 20, and then the second diagnosed on February 28—Bedford concluded that the two cases might have been linked.

This was the first time that sequencing data was widely used in a crisis to extend the traditional approach to epidemiology. Bedford helped pioneer the modern integration of these two disciplines. What he and others uncovered meant that COVID might have been spreading in Washington for at least six weeks. Somehow, the first traveler could have managed to infect someone who escaped detection and went on to transmit the virus to others. Bedford's genomic analysis, posted online on February 29, suggested that hundreds of people in Washington State might be already infected given the amount of time that had elapsed between the two cases.[16] By this calculus, the region was already facing a substantial outbreak, and nobody knew it.

This finding would be debated, and Bedford advanced later analysis that raised doubt about whether the two cases were actually connected, but even the possibility that coronavirus had been silently spreading in Seattle for almost six weeks was a dangerous turn of events. In fact, even if the two cases were not linked, we now know the virus had been spreading in American communities since at least January. It was hard to know, however, just how wide-

spread the infection was. During a critical six-week period over January and February, the Department of Health and Human Services had run into a series of challenges and committed blunders in its effort to roll out a diagnostic test that could screen for the coronavirus, leaving the country dangerously blind to its spread. Without a way to reliably test patients who had symptoms suggestive of SARS-CoV-2 infection, US doctors had no way to diagnose the sick or take measures to contain the spread.

One of the doctors on the front lines of fighting COVID, and trying to find ways to identify its spread, was Helen Chu, an infectious-disease specialist at the University of Washington School of Medicine. For months, Chu and her colleagues had been collecting nasal swabs from Seattle area residents, as part of the Seattle Flu Study, a novel project that they had launched with the support of the Gates Ventures, the venture capital arm of a vast public health investment enterprise maintained by Bill Gates and Melinda French Gates. (Bedford was also part of this effort.) Starting January 1, the research team had amassed more than 2,500 samples from people who had voluntarily enrolled in the study. The participants agreed to send in self-swabbed samples if they developed any signs or symptoms of flu. The goal was to see if this community-based approach could be an effective tool for identifying and observing the transmission of influenza. The researchers wanted to see if they could use this framework to develop a tripwire that would identify when flu was starting to spread—a "sentinel surveillance" program to screen for when the influenza virus was infiltrating a community. They were testing a new model for infectious disease surveillance.[17]

But Chu knew that the tools she had for screening participants for influenza, as well as the samples she had collected, could help determine whether coronavirus was also circulating in the Seattle area. After all, the swabs were from people who were reporting symptoms of a respiratory illness. If some of the participants who were sending in samples didn't have flu, perhaps they had COVID instead?

Chu's flu test relied on a process called polymerase chain reaction, or PCR. Using this approach, labs copy or "amplify" small segments of a virus's genetic material. The process uses primers and probes that are highly specific to the genetic sequences found in the particular virus that's being tested for.[18] These primers and probes will allow the test to render a positive result only if the probes are able to detect a precise snippet of RNA that's also found in the virus. This is how PCR is used to detect the presence of a particular virus and differentiate SARS-CoV-2 from more common coronaviruses. The technology can also determine how much of the virus is contained in a particular specimen, by quantifying the amount of RNA in a sample.

The key ingredient is heat. Throughout the process, RNA from the target virus is converted to cDNA (a single-stranded version of the double-helix DNA molecule), which is repeatedly warmed and cooled. With the help of special reagents, this thermal cycling triggers a chemical reaction that allows the primers to bind to the target stretch of cDNA and copy it. With each heating and cooling cycle, the number of copies of cDNA can be doubled. These DNA segments are complementary to RNA segments that are found in the virus. In just a few hours, and after thirty or forty cycles, there can be a billion or more copies of cDNA resulting from the original target piece of RNA. In a short time, PCR makes it possible to produce copious amounts of these complementary stretches of viral RNA that you're looking for, even starting with a very small amount of the actual virus.

The number of cycles needed to find a detectable level of virus is referred to as the cycle threshold, or Ct. Measuring this value can help determine how infectious a person is. If the machine needs fewer cycles in order to detect the viral RNA, it can mean there's more of the virus contained in the sample. The more virus someone is harboring, then in many cases, the more contagious they're likely to be. The number of cycles it takes to accumulate a detectable amount of coronavirus RNA is sometimes included in the results

sent to doctors, but not always, and many providers don't use this information to assess a patient's condition. They treat the test result as a binary outcome—either someone has coronavirus RNA present in their swab, and they're deemed to be infected, or the RNA cannot be found, and they're judged to be in the clear.

Another wrinkle is that the PCR machines are so sensitive, sometimes they can detect fragments of dead virus that have already been attacked by a patient's immune system and chopped into pieces. These dead fragments can linger for weeks after the patient has already beaten a viral infection. Even though the dead viral fragments can no longer cause disease, they can still register a positive result on the PCR machines. It's partly for this reason that the CDC eventually decided that they would no longer declare a patient to have an active infection based on the findings from the PCR tests alone. The CDC started to rely on a clinical criterion that also considered the number of days since a person's first COVID symptoms began.[19]

These limitations have stirred some criticism of PCR as a diagnostic tool.[20] Often, if a patient has any detectable virus by PCR, they're judged to be positive for coronavirus and are treated as if they're infectious. Sometimes labs will adjust the cycle thresholds needed to call a test positive and exclude results that are reached only by repeatedly cycling the machines, say forty or more times.[21] The challenge is that a high Ct value can mean that the patient has largely cleared the infection and is no longer contagious, but it can also mean that the person is newly infected, and the virus has just started to take its grip on them and hasn't had time to replicate itself widely.[22] So counting the number of cycles isn't always a reliable way to judge whether a person is indeed contagious.

Chu and her team had the right equipment to begin testing their samples for coronavirus. As a technical matter, it was straightforward. They would have to develop primers that were highly specific to stretches of RNA that are only found in the novel coronavirus's genome, strung among its thirty thousand base letters.[23] The research team was able to develop these primers and probes in January. A bigger challenge was to prove that the test was precise. Without a "positive control," an actual sample of the coronavirus, they couldn't fully verify that their test was able to accurately identify its RNA. To prove the performance characteristics of a diagnostic, researchers need access to samples of the coronavirus RNA to assess the new test. They could use inactivated copies of the actual virus, or synthetic genomic material for this purpose. But at this stage, Chu and her colleagues had neither. Only the CDC had those samples. Without them, there was no way to prove for certain that the homespun primers would bind to the actual virus. But the team at the University of Washington had a lot of experience in genomics and in developing tests for viruses and they were confident that their new COVID diagnostic tool was reliable.

There are a number of factors that combine to determine how valid a test is, but two of the most important parameters are the test's "sensitivity" and its "specificity."[24] A test's sensitivity is also called its true positive rate. It's a measure of the proportion of positive test results that are correctly identified. In other words, it's the percentage of people who have SARS-CoV-2 infection who are correctly diagnosed as being infected with the virus. A test's specificity is also called its true negative rate. It's a measure of the proportion of people who don't have the infection who are correctly identified as being virus free. A test with a low specificity would generate a higher number of false positives, saying that you were infected with the virus when you really weren't.[25] A test with a low sensitivity would generate a higher number of false negatives, saying that you don't have the infection when you really do.

To put this in practical terms, a test with a sensitivity of 80 percent may sound reliable, but in certain settings, it's not good enough to be practical. It means the test could miss one in five actual infections. If people are counting on that result to know whether they can go to work or visit with elderly relatives, a high rate of false negatives

can mean many people will engage in risky activities, where they can spread the virus to vulnerable contacts, and not know that they're infected. Likewise, a test with a specificity of 99 percent may similarly sound accurate. But again, its reliability may depend on how it's being used. Given that most people you test in a general population are not going to have SARS-CoV-2, a test with a 99 percent specificity means that a very high percentage of the people testing positive may be falsely positive. Consider this: let's say one in one hundred people you test actually has SARS-CoV-2 infection. That means the prevalence of the infection is 1 percent in the general population (which was the approximate prevalence during the epidemic in winter 2020). And let's say that a test has a 99 percent specificity, so for one hundred people you test, you will generate one false positive. That means that if you screen one hundred random people with such a diagnostic, you will get two positive results, on average. One of those positive results will be a person who is infected with the virus, and the other will be a false positive. So 50 percent of the positive results you generate will be false positives.

A lot of factors can affect a test's sensitivity and specificity. In a circumstance like COVID, where you are testing patients for a dangerous pathogen and trying to use testing as a way to control an outbreak, the reliability of the test is a critical factor. The FDA and the CDC asserted regulatory oversight for assuring its accuracy. This meant that if the scientists wanted to use their test on patients, they needed permission from the CDC and perhaps the FDA as well. The CDC has jurisdiction over tests that are being used for public health purposes as part of a pandemic response, and the FDA regulates tests that are used to diagnose individual patients for a disease. The Seattle researchers were in discussions with the CDC and Washington State health officials for weeks, seeking the ability to begin testing the samples they were receiving, to see if SARS-CoV-2 had arrived in the area. The debate with the CDC was initially over whether the University of Washington researchers would be allowed to use the test they had developed, or

whether they would have to send their samples to the CDC so the agency could do the testing in its reference lab in Atlanta. Chu and her colleagues finally got permission to go ahead and do the testing themselves in the University of Washington lab, so long as the testing was done for research purposes. This meant that if they found a positive sample, they wouldn't be able to report the result to the patient. But that wasn't practical. Chu and her colleagues knew that if they identified someone with an active SARS-CoV-2 infection they would want to share the results with the participant in order to help doctors identify cases and start tracking and tracing infected patients as a way to contain further spread.

Sharing the results with patients, however, would brand their test a medical diagnostic and in turn make it subject to FDA regulation. So they also sought permission from the FDA and the CDC to use the test for diagnosing patients.[26] But federal officials repeatedly rejected this idea, even as weeks went by and the pandemic worsened.[27] The CDC would only let the team test the samples as part of a research project. By February 25, Chu and her team decided to go forward and start using the test for "research use only," agreeing to the constraints under which federal regulators wanted them to operate.

They began by testing the samples collected most recently, and they got their first hit on February 27, on a nasal swab that had been sent to them just seventy-two hours earlier from a local high school student.[28] Because the sample was so recent, because the teenager might still be contagious, and because it involved a child, they felt an obligation to notify the patient and local health authorities. The case was independently confirmed by the Washington State Health Department the morning of February 28.[29] The school was promptly closed as a precaution. That same day, Chu's colleague Trevor Bedford joined the team to sequence the genome of the virus that had infected the boy; this was the sequencing that led Bedford to conclude that this new infection might be from the same viral lineage as the man diagnosed on January 20, who had been

identified as the first case to be found in the US.[30] If the two cases were linked, it meant that the virus had been spreading in the area for more than a month. Bedford and his research team immediately posted the sequence publicly to an online portal initially conceived to enable the global sharing of sequence information on influenza strains, called the Global Initiative on Sharing all Influenza Data (GISAID).[31]

Chu's effort showed how easy it might have been to authorize academic and commercial labs to start processing COVID tests on suspected cases and how hard it was for these labs to push forward on their own. The Seattle team notified this study participant without the CDC's agreement, and in response the CDC claimed that Chu and her team didn't have the appropriate patient consents in place and hadn't properly validated their test for diagnosing the participants. Once they notified the participant, as a regulatory matter, the test was no longer being used just for research, which made their effort subject to federal oversight. The CDC forced Chu and her team to pause what they were doing.

It didn't matter that the local ethics committee told them to report the results to the healthcare providers. And it didn't matter that the CDC had tried and, so far, failed to distribute its own diagnostic, leaving the nation dangerously devoid of a way to test for the novel coronavirus. To be sure, federal agencies like the FDA and the CDC were legally charged with ensuring the reliability of tests that were going to be used to screen patients for a novel pathogen like SARS-CoV-2, and the public health emergency made it even more critical that such tests be reliable. There were ways that the CDC could have given high-quality labs an easier path to advance tests in the setting of the crisis. But they jealously guarded this turf. When the team's lab reported its first positive hit, shortly thereafter the CDC told the lab to stop testing their samples.

Weeks went by before federal officials finally gave Chu and her colleagues a partial green light to resume their testing. Officials said the lab could test cases and report results on future samples

that they collected, so long as the lab used a new patient consent form that disclosed that the results might be shared with health officials. But for now, they couldn't test any of the 2,500 samples that had already been collected as part of their earlier flu study.[32] It wasn't until March 4, five days later, that an institutional review board that governs the conduct of trials involving patients determined that Chu could also proceed with testing the samples that had been collected in January and February as part of the flu study, with the results reported to public health authorities and patients.[33] By then, a lot of time had passed from when those swabs were first collected. If patients who were self-reporting symptoms of flu instead had the novel coronavirus, these infected individuals could have been circulating in the community for weeks.

Chu and her team tested samples dating back to January 1, again starting with the most recent samples. The first positive hits they got were on the samples collected in late February. In all, SARS-CoV-2 would be detected in 1.1 percent of the specimens that they screened, a whopping number considering that most health officials still maintained that the novel coronavirus wasn't yet circulating in the community. Many of these patients had reported respiratory symptoms and thought they might have the flu. Now, it turned out, some had COVID. The vast majority had reported mild illness and were treated at home. For most of them, it was probably too late to trace their contacts. So much time had passed from their initial infections that, if new cases were spawned by their illnesses, the subsequent patients were generations removed from the index cases. Two of the infected patients were children. Seven of the twenty-five patients who were identified with COVID reported being sick enough to seek medical care.[34]

It was a lot of positive hits to be getting on such a small and unselected pool of patients. If you were watching the evening news, the US was supposed to have just a dozen infections nationally. HHS officials were telling the White House staff that there was no widespread community transmission. The people in Chu's study

were not recent travelers from China or Italy. They were drawn from the general community. The novel coronavirus was spreading in Seattle.

The FDA would soon adopt a new strategy that would open the door for academic labs to begin testing patients and reporting the results back to their physicians. But in the meantime, Chu and her colleagues had run into some of the byzantine rules that limited the conduct of testing. The CDC exerted tight control over which labs could screen for COVID. And for now, the only facility in America that was authorized to do the testing was a single CDC lab in Atlanta.

It meant that doctors around the country had to forward samples to the CDC for testing. Physicians would first have to call the CDC and convince the agency to agree to take a sample for evaluation. It was a cumbersome process that limited the flow of specimens. By that point, there were far more people in America with unusual respiratory symptoms whose doctors wanted to test them, but the CDC's criteria still required that patients have all the signs and symptoms of COVID along with a history of travel to Wuhan or known exposure to someone who was already infected. This created a situation similar to when China's CDC required some contact with the Wuhan food market to be considered as a possible COVID case. It was only after Robert Redfield had suggested to George Gao that the Chinese CDC broaden its criteria that Gao revealed that the outbreak was probably out of control. Now, nearly two months later, the US CDC's requirements seriously narrowed who could qualify to be tested in America.[35] On March 1, the CDC's official tally of COVID cases rose from fifteen to seventy-five. Models developed later by researchers at Northeastern University show that by this date, the US probably had 28,000 infections.[36]

We had lost control. Without a widely available diagnostic test, we missed the chance to use case-based interventions—the ability to diagnose the sick, trace their contracts, and place people who had